

Author Index

Volume 17, 1999

- Ace, Christopher I., 241
Arcuri, Felice, 53
- Bagchi, Indrani C., 235
Boden, Guenther, 119
Broome, J., 197
Brudney, A., 257
Bulun, Serdar E., 349
Buster, John E., 327
- Carson, Daniel D., 217
Casson, Peter R., 327
Chang, Tara I., 153
Cintorino, Marcella, 53
Crawford, Sybil L., 299
Cummings, David E., 311
- Dhanasekaran, N., 167
Donnelly, K.M., 257
- Fazleabas, A.T., 257
Fleming, Honoree, 93
Fukuda, Michiko N., 229
Furman, Boris, 175
- Galan, Arancha, 267
Giudice, Linda C., 13
Glasser, Stanley R., 107
Gold, Leslie I., 73
Gravanis, Achille, 29
Greene, Michael F., 127
Guller, Seth, 39
- Harper, Andrew J., 327
Hausknecht, V., 3
- Homko, Carol J., 119
- Irwin, J.C., 13
- Jaffe, R.C., 257
Johannes, Catherine B., 299
- Kim, J.J., 257
Kirn-Safran, Catherine B., 217
Krikun, Graciela, 3, 45
Kumar, Nirmala S., 359
Kumar, Sushma, 235
- LaChapelle, Linda, 39
Leslie, Kimberly K., 359
Lessey, Bruce A., 197, 275
Lockwood, Charles J., 3, 45
Loeken, Mary R., 153
- Ma, Liang, 205
Maas, Richard L., 205
Margioris, Andrew N., 29
Martín, Julio Cesar, 267
Mazella, James, 23
Mazor, Moshe, 175
Merriam, George R., 311
Moe, Karen E., 339
Moley, Kelle H., 137
Murray, Michael J., 275
- Nozawa, Shiro, 229
- Okulicz, William C., 241
- Parekh, Trilok V., 73
- Pellicer, Antonio, 267
Pijnenborg, Robert, 63
- Reece, E. Albert, 118, 119, 167, 175, 183
Runic, R., 3
- Salafia, Carolyn M., 63
Santoro, Nanette, 297
Sasano, Hironobu, 349
Schatz, Frederick, 3, 45
Sestini, Silvia, 53
Sharpe-Timms, Kathy L., 107
Shea, Wendy, 197
Simón, Carlos, 267
Simpson, Evan R., 349
Sivan, Eyal, 119
Speroff, Leon, 1, 117, 195, 295
Srinivasan, S., 257
Starzyk, Kathryn A., 63
Stournaras, Christos, 29
- Tabibzadeh, S., 197
Tseng, Linda, 23
- Valbuena, Diana, 267
- Wang, E-Y., 3
Wiznitzer, Arnon, 175
Wu, Ying-King, 167
- Yao, Mylene, 205
- Zeitoun, Khaled, 349



Subject Index

Volume 17, 1999

- Abortion, spontaneous, 127–136
Adhesion, 267–274
Adhesion molecules, embryonic induction of in human EECs, 270–271
Adhesion phase
 embryonic regulation of, 270–273
 endometrial-embryonic interactions in, 268
Adipose tissue, 349–358
Adrenal cortex, 327–338
Adrenal hormone production, over various life stages, 328–330
Adrenal structure
 with aging, 328–330
 morphologic changes, 328
Adrenocortical function, therapeutic intervention, 330–333
Aging, 311–325, 339–348, 349–358
 adrenal structure with, 328–330
 adult GHD versus, 320–321
 GHD and normal, 313–315
AKT/protein kinase B, 169–170
Aldosterone, 328–329
Alpha-fetoprotein, 133–134
Alpha-2-macroglobulin, 93–106
Amphiregulin, 223
Androgens, 327–338
Angiogenesis, 275–290
Animal model, 243
Animal studies, in vivo, 140–143
Antiadhesion molecules, embryonic regulation of in human EECs, 271–272
Apoptosis, 137–151, 153–165, 267–274
 FasL-mediated, 39–40
 reduction in critical progenitor cells by, 155–156
Apposition phase
 embryonic regulation of, 269–270
 endometrial-embryonic interactions in, 268
Arachidonic acid, 167–174
Aromatase, 349–358
Aromatase expression
 in adipose tissue and breast cancer, 351–353
 in bone, 354
 in brain, 354–355
 in endometrial cancer, 355–357
 in postmenopausal endometriosis, 355
 in skin, 353–354
Biochemical differentiation, in cycling endometrium, 94
Biotin, 93–106
Blastocyst, 137–151, 197–203
 apoptosis, glucose-induced, 146–148
 coordinated regulation of endometrial epithelial apoptosis induced by, 272–273
 implantation, 31–33, 107–115
Blood supply, tumor recruitment of new, 282–283
BMPs, *see* Bone morphogenetic proteins
Body temperature, 339–348
Bone, 349–358
Bone morphogenetic proteins (BMPs), 74
Brain, 349–358
Breast, 359–370
Breast cancer, 349–358, 360–365
Bystin, 229–234
 expression of in cells involved in implantation, 233
 interaction of with trophinin and tasin, 232
Calcitonin, 238
Cancer, 93–106, 275–290, 359–370, *see also* specific types
 alterations of TGF- β receptors in human, 76–77
 human, 73–92
 incidence of and deaths from among American women, 359–360
 progression, matrix metalloproteinases and, 281–282
Carbohydrate metabolism, 119–125
Cell adhesion molecules, 279–280, 281
Cell culture, 93–106
 uses for, 101–103
 varying microenvironments in, 99–101
Cell cycle
 genes/proteins, 80
 regulation, 78–80
Cell interactions, 103
Cell paradigms, transformed and transfected, 110
Central nervous system actions, of estrogen, 345
Chemokines, embryonic regulation of in human EECs, 269–270
Chorionic gonadotropin, 17–18, 260–261
Circadian rhythm, 339–348
Cloning, 229–234, 247–250
Colorectal cancer, 367
Congenital abnormalities, 127–136
Congenital malformations, 127–136
 diagnosis of, 133–135
 mechanism for, 157–159
Corticotropin-releasing hormone (CRH), 29–38
Cortisol, 329
Cortisol/DHEA
 cardiovascular risks and, 333–334
 cognition, memory, and mood and, 335
 glucose tolerance and, 334
 immune augmentation and, 334–335
 ratio, 329–330
COX-1, *see* Cyclooxygenase-1
COX-2, *see* Cyclooxygenase-2
CRH, *see* Corticotropin-releasing hormone
Cyclooxygenase-1 (COX-1), 171–172
Cyclooxygenase-2 (COX-2), 167–174
Cytokines, 17–18

- Decidua, 13–21, 53–61
- Decidualization (DZ), 3–12, 29–38
- association of with endometrial hemostasis, 46
 - association of with enhanced TF and PAI-1 expression, 47
 - cultured human endometrial stromal cells as model for, 47–48
 - induction of stromal, 33
- Dehydroepiandrosterone (DHEA), 327–338
- Dehydroepiandrosterone administration
- with concomitant estrogen replacement therapy, 333
 - dosing, 330–332
 - pharmacokinetics, 330–333
 - potential side effects, 333
 - routes, 332–333
- Dehydroepiandrosterone sulfate (DHEAS), 327–338
- Developmental control genes, interference with expression of essential, 156–157
- DHEA, *see* Dehydroepiandrosterone
- DHEAS, *see* Dehydroepiandrosterone sulfate
- Diabetes, 153–165, 183–194
- disruption of embryonic development by maternal, 154–159
 - mellitus, 127–136
 - preimplantation development, 140–148
 - related malformation gene, 155
 - toxic effects of maternal, 157–159
- Diabetic embryopathy, 167–174, 175–181
- metabolic cause of, 154
 - prevention of, 190–193
- Diabetic mutagenesis, 155
- Differential display analysis, 250–252
- Differentiation, 93–106
- altering of protein regulation by, 98–99
 - biochemical, 94
 - epithelial, 95
 - morphological, 94–95
 - using endometrial epithelial cell lines to study, 95–96
- Domes, 93–106
- DZ, *see* Decidualization
- ECA, *see* Endometrial cancer
- ECM, *see* Extracellular matrix
- EECs, *see* Endometrial epithelial cells
- EGF family, *see* Epidermal growth factor family
- Eicosanoids, biosynthesis, degradation, and action of, 176–177
- Embryo, 153–165, 267–274
- development, disruption of by maternal diabetes, 154–159
 - gene expression, molecular mechanisms by which maternal diabetes perturbs, 159–161
 - implantation, 276–277, 282–285
 - studies, in vivo, 143
- Endogenous opioid peptide (EOP), 30
- Endometrial cancer (ECA), 80, 349–358, 365–366
- Endometrial carcinoma, role for TGF- β in pathogenesis of, 82–86
- Endometrial complementary DNA populations, preparation of, 243–244
- Endometrial-embryonic interactions, in vitro model for, 268
- Endometrial epithelial cells (EECs), 267
- embryonic induction of adhesion molecules in human, 270–271
 - embryonic regulation of antiadhesion molecules in human, 271–272
 - embryonic regulation of chemokines in human, 269–270
- Endometrial epithelial and stromal cell models, contention, 109–110
- Endometrial hemostasis, association of decidualization with, 46
- Endometrial hyperplasia and carcinoma, TGF- β -related mechanisms of loss of growth regulation in, 80–82
- Endometrial neuropeptides, paracrine actions of, 31
- Endometrial proteins/glycoproteins, secretion of stage specific, 110–112
- Endometrial receptivity, embryonic regulation of, 268–269
- Endometrial stromal/decidual cells, regulation of prolactin secretion of human, 24
- Endometrial vascular system, regulation of, 33–35
- Endometriosis, 93–106, 349–358
- Endometrium, 3–12, 13–21, 29–38, 93–106, 197–203, 235–240, 241–255, 257–265, 267–274, 359–370
- biochemical differentiation in cycling, 94
 - human, 23–27
 - IGF family in cycling, 14–15
 - morphological differentiation in cycling, 94–95
 - regulation of IGFBP-1 in, 17
 - structure of nonhuman primate, 242–243
- Endothelial cells, proliferation of, 284–285
- EOP, *see* Endogenous opioid peptide
- Epidermal growth factor (EGF) family, 236–237
- Epithelial cells, polarized, 112–114
- Epithelial differentiation, using primary cultures to study, 95
- Epithelium, 93–106
- ER, *see* Estrogen receptor
- EREs, *see* Estrogen-responsive elements
- ERK, *see* Extracellular signal regulated kinase
- Estrogen, 205–216, 339–348, 349–358, 359–370
- central nervous system actions of, 345
 - role of, 258–260
 - stimulation of prolactin receptor by, 25–26
- Estrogen receptor (ER), 81
- Estrogen-responsive elements (EREs), 31, 49
- Extracellular matrix (ECM), 4, 277, 280–281, 284–285
- Extracellular signal regulated kinase (ERK), 167–174
- FasL, *see* Fas ligand
- FasL expression, in human placenta, 40–41
- Fas ligand (FasL), 39
- Fetal immune privilege, model of, 40–41
- Final menstrual period (FMP), 300, 301
- First trimester events
- metabolic control and, 127–132
 - relationship between control and, 130–132
- FMP, *see* Final menstrual period
- Free oxygen radicals, 175–181, 183–194
- Free radicals, excess, 187–189
- Fuel metabolism
- during early pregnancy, 119–120
 - during late pregnancy, 120–123
- Gene
- activation, 77–78
 - analysis of important known, 244–246
 - P-dependent, 246–250
 - regulation, 241–255, 257–265
- Gestational diabetes, 119–125
- GH, *see* Growth hormone
- GHD, *see* Growth hormone deficiency
- GHRH, *see* Growth hormone-releasing hormone
- Glioblastoma, 367
- Glucocorticoid metabolism, 53–61
- Glucocorticoid response element (GRE), 31
- Glucose, 153–165
- induced blastocyst apoptosis, 146–148

- potential signal transduction pathways mediating genotoxic effects of, 160–161
 role of in mediating genotoxic effects of diabetic pregnancy, 160
 transport, 137–151
 utilization studies, 143–146
- Glycoconjugate expression, control of, 219
- Gonadal steroids, 73–92
- GRE, *see* Glucocorticoid response element
- Growth factors, 17–18
- Growth hormone (GH), 311–325
 action, sex steroid effects on, 315–316
 dosing, recommended, 320
 secretagogues, 322–323
 therapy, 319–320, 322
 treatment, 318–319, 323
- Growth hormone deficiency (GHD), 311–325
 adults, benefits of GH treatment in, 318–319
 aging versus adult, 320–321
 diagnosing adult, 321
 model of adult, 316–320
 normal aging and, 313–315
 similarities and differences between aging and adult, 320–321
 syndrome, adult, 316–318
- Growth hormone-releasing hormone (GHRH), 311–325
- Growth hormone secretion
 consequences of reduced, 316–320
 mechanisms of age-related changes in, 312–316
 regulation of, 312–313
 sex steroid effects on, 315
- Growth inhibition, 73–92
- HB-EGF, *see* Heparin-binding epidermal growth factor-like protein
- Hemostasis
 association of decidualization with endometrial, 46
 mediation of by tissue factor and plasminogen activator inhibitor, 46–47
- Heparin-binding epidermal growth factor-like protein (HB-EGF), 223
- Heparin sulfate proteoglycans (HSPGs), 221
- HESCs, *see* Human endometrial stromal cells
- HIP, 221–223
- HIP/L29, 217–227
- Homeobox genes, in reproduction, 211–212
- Hormone replacement therapy (HRT), 81
- Hormone response elements (HREs), 241
- Hormones, in tumor initiation and growth, 360–367
- Hox*, 205–216
 genes and roles, in reproduction, 207–213
 proteins, functions of in implantation, 212–213
- Hoxa-10* functions, in maternal uterus, 208–209
- HREs, *see* Hormone response elements
- HRT, *see* Hormone replacement therapy
- 11 β -HSD, *see* 11 β -Hydroxysteroid dehydrogenase
- HSPGs, *see* Heparin sulfate proteoglycans
- Human cancers, 73–92
 aberrant TGF- β -related cell cycle genes/proteins in, 80
 alterations in TGF- β receptors in, 76–77
 disruption of TGF- β signal transduction in, 78
- Human endometrial stromal cells (HESCs), 4
 expression of MMP and TIMP during in vitro decidualization of, 6
 expression of PAs during in vitro DZ of, 4–5
 induction of prolactin in, 24–25
 as model for decidualization, 47–48
- steroid withdrawal effects on PA, MMP, and TIMP1 expression in cultured, 6–8
 TF and PAI-1 expression, molecular mechanisms for progesterone-induced, 48–49
- Human, 197–203
- 11 β -Hydroxysteroid dehydrogenase (11 β -HSD), 53–54
 physiological functions of, 54
 placental, 54–55
- Hyperglycemia, 175–181, 185
- Hypoglycemia, 167–174
- Hypoxia, 18
- IDMs, *see* Infants of diabetic mothers
- IGF, *see* Insulinlike growth factor
- Implantation, 13–21, 29–38, 197–203, 205–216, 217–227, 229–234, 235–240, 267–274, 275–290
 blastocyst, 31–33, 107–115
 embryo, 276–277, 282–285
 expression of steroid-regulated genes during, 236–238
 expression of trophinin, bystin, and tatin in cells involved in, 233
 general changes in cell surfaces during, 218–219
 hormonal regulation of *AbdB Hoxa* genes during, 209–211
 human cell lines useful for studying, 230–231
 in human and mouse, 206
 IGFBP-1 and, 16, 18
 possible functions of *Hox* proteins in, 212–213
 provisional model for genetic control of, 213–215
 research, experimental methods used in, 206–207
 steroid hormone regulation of embryo, 236
 window, 198
- Infants of diabetic mothers (IDMs), 127
- Infertility, 197–203
 causes of, 198
 molecular lesions in, 198–201
 myo-Inositol metabolism, abnormalities in, 189–190
- Insulinlike growth factor (IGF), 13–21
 binding protein, 13–21
 stimulation of prolactin receptor by, 25–26
- Insulinlike growth factor family, 13–14
 in cycling endometrium, 14–15
 at decidual, 15–16
- Insulin resistance, 119–125
- Integrins, 111–112, 275–290
 $\alpha_v\beta_3$ Integrins, 223–224
- Invasion, 275–290
- Ishikawa cells, 93–106
- JNK, *see* Jun N-terminal kinase
- Jun N-terminal kinase (JNK), 167–174
- Latent TGF- β (LTGF- β), 75
- Leukemia inhibitory factor (LIF), 93–106, 236
- LIF, *see* Leukemia inhibitory factor
- Lipid metabolism, 119–125
- LTGF- β , *see* Latent TGF- β
- Malformation, 183–194
 gene, diabetes-related, 155
 major, 127–130, 132
 range of, 154
- Malignant melanoma, 367
- Matrix metalloproteinases (MMPs), 3–12, 275–290
- Maturation, 241–255, 257–265
- Melanoma, malignant, 367
- Menopause, 299–309
- Menopause-related menstrual cycle changes, 303–306

- Menses, 93–106
 Menses and regeneration, 102
 Menstrual bleeding
 age-related hormonal changes prior to changes in, 300–301
 relation between perimenopausal hormone profiles and, 307–308
 Menstrual cycle, 299–309
 changes, menopause-related, 303–306
 changes, in perimenopause, 302–307
 length, age-related changes in, 303
 variability, age-related changes in, 303
 Menstrual cyclicity, changes occurring after first break in, 301
 Menstrual flow, changes in, 306–307
 Migration, cellular elements of, 277–280
 MIS, *see* Mullerian inhibitory substance
 MMPs, *see* Matrix metalloproteinases
 Models, 107–115
 endometrial epithelial and stromal cell, 109–110
 in vitro, 112–114
 Molecular markers, 235–240
 Morphological differentiation, in cycling endometrium, 94–95
 Morphologies, extending culture conditions to effect different, 96–98
 Muc-1, *see* Mucin-1
 Mucin, 217–227
 Mucin-1 (Muc-1), 111, 237–238
 Mullerian inhibitory substance (MIS), 74
 Mutagenesis, diabetic, 155
 Myometrial contractility, regulation of, 35
- Neural tube, 153–165
 Neuropeptides, paracrine actions of endometrial, 31
 Nonlymphoid cells, role of FasL immunosuppression in, 40
- ODNs, *see* Oligodeoxynucleotides
 Oligodeoxynucleotides (ODNs), 238
 Oncogenesis, role of transforming growth factor- β in, 74
 Opioids, 29–38
 Osteoporosis, 349–358
 Ovarian cancer, 366–367
- PA, *see* Plasminogen activator
 PAI, *see* Plasminogen activator inhibitor
 Pathogenesis
 clinical, 184
 experimental, 185–190
 Pax-3, 153–165
 Peptides, insulinlike, 17
 Perimenopausal hormone profiles, relation between menstrual bleeding and, 307–308
 Perimenopausal variability, in hormone concentrations, 301–302
 Perimenopause, 299–309, 339–340
 hormonal changes during, 300–302
 menstrual cycle changes in, 302–307
 Phosphatidylinositol-3-kinase, 169–170
 Phospholipase A₂ (PLA₂), 167–174
 Pituitary, 311–325
 PKC, *see* Protein kinase C
 PLA₂, *see* Phospholipase A₂
 Placenta, 53–61, 275–290
 FasL expression in human, 40–41
 immune privilege of, 40
 Placental physiology, systemic vascular risk factors and, 68–69
- Plasminogen activator (PA), 3–12
 expression of during in vitro DZ of HESCs, 4–5
 inhibitor (PAI), 47–47
 p38MAPK, *see* p38-mitogen-activated kinase
 p38-mitogen-activated kinase (p38MAPK), 167–174
 Postmenopausal women, older, 340
 Postmenopause, early, 339–340
 Post-receptor signal transduction, 77–78
 PRE, *see* Progesterin response element
 Pregnancy, 13–21, 119–125, 127–136
 complications, 127–136
 fuel metabolism during early, 119–120
 fuel metabolism during late, 120–123
 function of uteroplacental vasculature in complicated, 67–68
 function of uteroplacental vasculature in early, 65–66
 IGFBP-1 in human, 18
 prostaglandins in diabetic, 178–180
 role of glucose in mediating genotoxic effects of diabetic, 160
 structural pathology of uteroplacental vasculature in complicated, 66–67
 structure of uteroplacental vasculature in early, 64–65
 Preimplantation development, diabetes, 140–148
 Preimplantation period, 138–140
 metabolism, 139–140
 morphologic changes, 138
 Prevention, 183–194
 Primary cultures, 73–92
 PRL, *see* Prolactin
 PRL-R, *see* Prolactin receptor
 Progesterone, 205–216, 241–255, 257–265, 342, 359–370
 Progesterin, stimulation of prolactin receptor by, 25–26
 Progesterin response element (PRE), 49
 Prolactin (PRL), 23–27
 gene promoter, expression of in endometrial stromal/decidual cells, 24–25
 induction of, 24–25
 mitogenic effect of, 26
 physiological function of in maternal-fetal unit, 26
 promoter, 26
 regulation of amniotic osmolality by, 26
 secretion, regulation of, 24–25
 Prolactin receptor (PRL-R), 23
 Prostaglandins, 167–174, 178–180, 183–194
 Prostanoids, 175–181
 Protein kinase C (PKC), 167–174
 Protein regulation, altering of by differentiation, 98–99
 Proteins/glycoproteins, secretion of stage specific endometrial, 110–112
 Proteoglycan, 217–227
- Reactive oxygen intermediates, 161
 Reactive oxygen species (ROS), 167–174
 Receptivity, 197–203
 Receptor, 23–27, 359–370
 Reproduction, homeobox genes in, 211–212
 Reproductive hormones, 299–309, 341–342
 Rhesus monkey, 241–255, 257–265
 ROS, *see* Reactive oxygen species
- Sequencing, of P-dependent genes, 247–250
 Sex steroid
 effects of on GH action, 315–316
 effects of on GH secretion, 315
 Signaling paradigm, 172–173
 Signal transduction

- disruption of TGF- β , in human cancers, 78
- post-receptor, 77-78
- Skin, 349-358
- Sleep, 339-348
 - difficulties, characteristics of in older women, 340-341
 - disruptions, vasomotor symptoms associated with, 342-343
 - disturbance, prevalence of in older women, 339-340
 - effects, possible mechanisms of, 342-345
 - reproductive hormones and, in older women, 341-342
- Somatopause, treatment of, 321-323
- Somatotropin, 311-325
- Spontaneous abortions, 127-136
- Steroid
 - agonists and antagonists, 101-102
 - gonadal, 73-92
 - hormones, 235-240
- Steroidogenesis, 349-358
- Stress reactivity, 344
- Stromal/epithelial interactions, importance of, 85-86
- Subtractive hybridization, 246-247
- Tastin, 229-234
 - expression of in cells involved in implantation, 233
 - interaction of bystin with, 232
- TF, *see* Tissue factor
- TGF- β , *see* Transforming growth factor- β
- TIMP, *see* Tissue inhibitor of matrix metalloproteinase
- Tissue factor (TF), 46-47
- Tissue inhibitor of matrix metalloproteinase (TIMP), 4
- Transcription factors, 205-216
- Transforming growth factor- β (TGF- β), 74
 - background on, 74-75
 - cell cycle regulation by, 78-80
 - receptors, 76-77
 - role of in oncogenesis, 74
 - role for in pathogenesis of endometrial carcinoma, 82-86
- Trophinin, 229-234
 - expression of in cells involved in implantation, 233
 - identification of, 231-232
 - interaction of bystin with, 232
 - in vivo role of, 233-234
- Trophoblast, 53-61
 - expression of FasL by, 41-42
 - FasL, potential lymphocyte targets of, 42-43
 - interface, 15-16
 - role of, 261-262
- Tumor
 - angiogenesis, 282-285
 - growth and metastasis, 280-282
 - immune privilege of, 40
 - initiation and growth, hormones in, 360-367
 - neovascularization, 282-283
 - progression, 282-285
- Ultrasound examination, 134-135
- Uterine antiadhesive molecules, loss of, 219-221
- Uterine corticotropin-releasing hormone, 30-31
- Uterine luminal epithelial, morphological alterations in, 108-109
- Uterine opioid peptides, 30-31
- Uterine receptivity, 107-115, 235-236
 - establishment of, 258-262
 - laboratory strategies for study of, 109
 - markers of, 238
- Uterine vasculature, nonpregnant, 63-64
- Uteroplacental vasculature, 64-68
- Uterus, 217-227
- Yolk sac injury, 185-187